

Managing Infection and Sepsis in Trauma Patients

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KEY WORDS

- trauma
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- systemic inflammatory response syndrome (SIRS)

Systemic inflammatory complications of trauma can result from both contaminated and uncontaminated trauma. Contaminated trauma refers to tissue damage that is associated with known bacterial inoculation; classic examples include bite wounds, gunshot wounds, lacerations or degloving injuries contaminated with organic material, ruptured bowel, and any wound containing more than 100,000 bacteria per gram of tissue. Failure to control contamination can lead to systemic infection and sepsis. Uncontaminated trauma is usually associated with blunt trauma with no external source of infection; however, systemic inflammatory response and/or sepsis can result from activation of the inflammatory cascade and bacterial or endotoxin translocation from the gastrointestinal tract. Severe tissue damage can produce a systemic inflammatory response even in the absence of obvious infection.

The following terms were defined by the American College of Chest Physicians and Society of Critical Care Medicine^{1,2}:

- *sepsis*—systemic inflammatory response to infection
- *systemic inflammatory response syndrome (SIRS)*—systemic, hypermetabolic inflammatory response that does not require evidence of infection (e.g., severe tissue trauma or cancer can initiate such an inflammatory response with no evidence of infectious agents)
- *septic shock*—sepsis with hypotension in the face of appropriate fluid therapy
- *multiple organ dysfunction system (MODS)*—progressive, serial loss of organ function

CONTAMINATED TRAUMA

General Wound Management

Failure to control local contamination and maintain hemodynamics may lead to systemic infection and sepsis. The goals of emergency wound management are to preserve function and prevent infection. Primary management must always address the animal's life-threatening problems (e.g., airway, breathing, circulation, and neurologic dysfunction) prior to definitive wound management. Once the animal is stable, the wound should be assessed. Bleeding should be controlled using direct pressure and elevation if possible. If direct pressure is inadequate for a distal limb wound, a blood pressure cuff can be placed proximal to the wound and inflated to a level slightly greater than the patient's systolic pressure. This can remain in place up to 2 hours without causing injury to nerves and other structures.³ The wound is contaminated if it is more than 3 to 4 hours old, contains pus, feces, or soil, was caused by a compressive force resulting in local disturbance of blood flow, or is located in a region of high bacterial population or poor blood flow.

Wounds that are initially "clean" can become contaminated from the bacteria introduced on clipper blades, the clinician's hands, or spray from the clinician's mouth. Bacterial concentrations of more than 100,000 per gram of tissue result in wound infection.^{3,4} Lower densities of bacteria can cause infection if the patient's immune function is impaired (e.g., by soil contaminants, immunosuppressive drugs such as steroids, or underlying disease).³ Wound preparation should decrease the local bacterial population without interfering with the natural host defenses. The intact skin surrounding a wound—not the wound itself—should be gently but thoroughly scrubbed with an iodophor or chlorhexidine solution.³ Contaminated wounds should be lavaged with copious amounts of sterile saline; irrigation should be performed using pressure generated by an 18 to 19 gauge needle or catheter and a 35 to 60 ml syringe.⁵ Noncontaminated wounds can be lavaged with low pressure. A rule of thumb for choice of lavage fluid is "the only solution that should be placed in a wound is one that can be safely poured in the physician's (veterinarian's) eye."⁶

Debridement is necessary to remove dead and devitalized tissues, which can harbor bacteria and interfere with the natural immune response.³ Infected wounds should not be sutured; if, however, sutures are necessary in a contaminated or infected wound, the fewest

and smallest possible sutures should be placed with minimal trauma and tension. Synthetic monofilament sutures are preferred over natural fiber multifilament ones. In wounds requiring temporary support, absorbable sutures can be used; nonabsorbable sutures are preferred for long-term support.^{3,7} The use of drains in wounds provides a mechanism for removal of infection; however, it also allows an avenue for bacterial contamination and tissue inflammation. Drains are helpful in draining abscesses and eliminating dead space.^{3,8} An additional means of eliminating dead space and protecting the wound is through bandaging.⁹ Experimentally, antibiotics are most beneficial when given prior to injury; however, this is not practical in the clinical setting. The decreased local blood flow that accompanies the injury results in decreased antibiotic delivery. If the wound is contaminated or infected, antibiotics of the appropriate spectrum and with the ability to penetrate the wound should be started early in the treatment.^{4,10} Steroids are contraindicated in animals with severe wounds as they interfere with both wound healing and the immune response, making wounds more susceptible to infection.¹¹

Gunshot Wounds

Gunshot wounds are a problem in both urban and rural societies in America. The kinetic energy of a projectile (i.e., $[\text{mass} \times \text{velocity}^2]/2$) influences the amount of tissue injury. Bullet wounds cause shear damage and compression damage to tissues. As the energy is dispersed throughout the tissue, it creates shock waves that further distribute the energy and damage. In addition to the kinetic energy and orientation of the projectile, the amount of damage sustained by a given tissue depends on the tissue's elasticity and density.³ Bone and liver sustain a greater degree of damage than does lung or fat.¹¹ The shock waves can result in avulsion of nerves and vessels and can rupture the intestine. The potential for extensive direct and indirect damage has led to the recommendation that bullet wounds to the abdomen be explored.¹² This recommendation is supported by a retrospective study of gunshot wounds in veterinary patients.¹³ All gunshot wounds are considered contaminated. The projectile can carry bacterial contaminants from the air and skin into deeper tissues. Projectiles also cause cavitation lesions, which produce a negative pressure that sucks surface contaminants into the wound. The shock waves and local compressive forces decrease blood flow and interfere with normal immune clearance of bacteria. Minor or superficial wounds with minimal tissue trauma should be treated as contami-

nated wounds. Penetrating wounds should be explored to remove foreign material and devitalized tissue, either of which may act as a nidus for infection and inflammation.

Bite Wounds

Bite wounds are also compression-type wounds. There is a small amount of shear injury seen at the puncture site, but the majority of the damage is hidden in deeper tissue planes. The oral cavity contains anaerobic and aerobic bacteria. These wounds are generally infected. Involvement of deep tissues or bone or penetration into the thorax or abdomen is an indication for irrigation, surgical debridement, and exploration of the wound. Failure to provide adequate debridement and drainage of such wounds can lead to bacteremia, SIRS, and death. This appears to be most common in cases in which the wounds are sutured but adequate drainage is not provided. The use of drains or open wound management may prevent some of the septic complications. The use of antibiotics does not eliminate the need for adequate wound management.

The management goals for contaminated wounds are to stabilize the patient and control infection and inflammation. Failure to do so can lead to systemic involvement, SIRS, multiple organ dysfunction, and death.

SEPSIS

An animal with a local infection or tissue injury will develop an inflammatory response initiated primarily by the white blood cells and immune system. The function of this response is to increase blood flow to the area and deliver cells and agents that can battle the infection or eliminate the tissue injury. These animals have local areas of heat, pain, redness, and swelling, the hallmark signs of inflammation. The appropriate response is to keep the inflammation localized. The development of a systemic response can result from excessive tissue trauma, inability to control the local infection, remote injury associated with hypotension or hypoxia, or a secondary insult (e.g., aspiration pneumonia).

The development of SIRS is associated with signs of tachycardia, hyperemic mucous membranes, rapid capillary refill, and hypoglycemia. Other common findings include either an increase or decrease in rectal temperature and tachypnea. These effects are mediated by the systemic release of inflammatory mediators including cytokines, prostaglandins, and nitric oxide. The net result is that tissues do not get adequate blood flow, oxygen, or nutrients and accumulate metabolic by-products. In addition, tissues and organs

are directly damaged by activated inflammatory cells. This can lead to further activation of the inflammatory cascade, impaired perfusion, release of cytotoxic mediators, and, finally, organ failure and death (for a review of the mediators of SIRS, see Davies and Hagen¹⁴).

Recognition of septic shock or SIRS requires an index of suspicion. Animals at risk are those with an identified source of infection such as severe or infected wounds (especially bite wounds), extensive trauma, severe inflammatory diseases such as pancreatitis, a history of prolonged hypotension, extensive surgery, or trauma. These cases should be monitored closely for development of hypoxia, hypotension, or other secondary insults that may amplify the inflammatory response. Aggressive management is necessary to minimize the risks of SIRS and MODS. The source of inflammation should be removed if possible; infections should be controlled; and perfusion and oxygenation should be maintained. The development of SIRS is frequently accompanied by progressive organ failure. The clinical signs are a reflection of the organ affected. The cardiovascular and coagulation systems are often affected early in the course of the syndrome. Blood vessels dilate, resulting in red mucous membranes and distributive shock. The heart may develop arrhythmias and decreased contractility. Coagulation is activated, leading to consumption of clotting factors and platelets. Clinically, patients may develop petechia or hemorrhage. Renal function is compromised, and urine production falls. The gastrointestinal tract is prone to ischemia in states of poor perfusion; the ischemic intestine predisposes to bacterial translocation, which leads to more inflammation. Animals may develop diarrhea or melena. The central nervous system can be affected by hypoglycemia or products of activated inflammatory cells. Clinically, animals may become stuporous or have seizures. The lungs may

accumulate fluid and neutrophils, leading to a condition called adult respiratory distress syndrome (ARDS). This syndrome is characterized by hypoxia, hypercarbia, and the requirement for positive pressure ventilation (for reviews, see References 2, 15, and 16).

Mortality in humans that develop SIRS is 40% to 60%.¹⁴ It is apparent that veterinary patients do not fare any better. Early recognition of septic shock or SIRS is essential, but the best treatment is prevention. Once an animal develops septic shock or SIRS, treatment is primarily supportive.

REFERENCES

1. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. *Crit Care Med* 20:864–874, 1992.
2. Beal AL, Cerra FB: Multiple organ failure syndrome in the 1990s. Systemic inflammatory response and organ dysfunction. *JAMA* 271:226–233, 1994.
3. Edlich RF, Rodeheaver GT, Morgan RF, et al: Principles of emergency wound management. *Ann Emerg Med* 17:1284–1302, 1988.
4. Robson MC: Disturbances of wound healing. *Ann Emerg Med* 17:1274–1278, 1988.
5. Stevenson TR, Thacker TG, Rodeheaver GT, et al: Cleansing traumatic wound by high pressure syringe irrigation. *J Am Coll Emerg Phys* 5:17–21, 1976.
6. Branemark PI, Albrektsson B, Lindstrom J, et al: Local tissue effects of wound disinfectants. *Acta Chir Scand* 357:166–176, 1966.
7. Smeak DD, Wendelburg KL: Choosing suture materials for use in contaminated or infected wounds. *Compend Contin Educ Pract Vet* 11:467–475, 1989.
8. Lee AH, Swaim SF, Henderson RA: Surgical drainage. *Compend Contin Educ Pract Vet* 8:94–103, 1986.
9. Swaim SF, Wilhalf D: The physics, physiology and chemistry of bandaging open wounds. *Compend Contin Educ Pract Vet* 7:146–156, 1985.
10. Hunt TK: The physiology of wound healing. *Ann Emerg Med* 17:1265–1273, 1988.
11. Trott A: Mechanisms of surface soft tissue trauma. *Ann Emerg Med* 17:1279–1283, 1988.
12. McCarthy MC, Lowdermilk GA, Canal DF, et al: Prediction of injury caused by penetrating wounds to the abdomen, flank, and back. *Arch Surg* 126:962–965, 1991.
13. Fullington RJ, Otto CM: Characteristics and management of gunshot wounds in dogs and cats: 84 cases (1986–1995). *JAVMA* 210:658–662, 1997.
14. Davies MG, Hagen PO: Systemic inflammatory response syndrome. *Br J Surg* 84:920–935, 1997.
15. Rackow EC, Astiz ME: Pathophysiology and treatment of septic shock. *JAMA* 266:548–554, 1991.
16. Bone RC, Grodzin CJ, Balk RA: Sepsis: A new hypothesis for pathogenesis of the disease process. *Chest* 112:235–243, 1997.

